Exhibit 25

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February 16, 2018

Via E-mail and Priority Mail
All Counsel (See enclosed service rider)

Re: *Rimondi, et al. v. BASF Catalysts LLC, et al.* (MID-L-02912-17)
Jacqueline Moline, M.D., MSc, FACP, FACOEM's Expert Report

Counselors:

Enclosed please find a copy of the report of Jacqueline Moline, M.D., MSc, FACP, FACOEM in the above-referenced matter.

If you have any questions or require any additional information, please contact Emily Kortright at 212-421-2800.

Regards,

Joseph N. Cotilletta

Encl.

Northwell Health
Occupational Medicine, Epidemiology and Prevention

February 8, 2018

Joseph Cotilletta, Esq.
The Lanier Law Firm
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New York, NY 10022

Re: Ricardo Rimondi

Dear Mr. Cotilletta:

I am writing to report the results of my evaluation of the materials listed below pertaining to Mr. Ricardo Rimondi. I also had the pleasure of evaluating him on November 9, 2017. I have reviewed these materials in the context of my pre-existing knowledge, training, and experience in the field of occupational medicine. These materials are of the type I and other specialists in occupational medicine normally rely upon and are sufficient to form a reliable basis for my opinions contained within this report. All of the opinions stated in this report are given within a reasonable degree of medical certainty.

This report and the opinions stated in the report are based on the listed materials and my 25 years of training, education, and experience in the area of asbestos-related occupational medicine. Over the past 25 plus years, I have had the opportunity to evaluate and treat hundreds of patients with asbestos exposure, many of whom have asbestos related diseases.

Qualifications:

I am a physician licensed in the State of New York, specializing in the field of occupational and environmental disease. I have been a practicing physician since I graduated from medical school in 1988.

I attended the University of Chicago and received a Bachelor of Arts degree with Honors, with a major of History, Philosophy, and Social Studies of Science and Medicine. I then continued at the University of Chicago – Pritzker School of Medicine, where I obtained my medical degree in 1988. I was elected to the Alpha Omega Alpha Honor

Society, and was also awarded an American Medical Women's Association Award. Following medical school graduation, I was an intern and resident in Internal Medicine at Yale University – Yale New Haven Hospital from 1988 – 1991. Upon completion of my Internal Medicine Residency program, I completed a second residency at the Mount Sinai School of Medicine in Occupational Medicine, from 1991 – 1993. During my Occupational Medicine Residency Program, I obtained my Master of Science Degree in Community Medicine (equivalent degree to a Masters of Public Health) in 1993. I began to evaluate dozens of patients with asbestos exposure during my residency program at Mount Sinai. I am board certified in Occupational Medicine and in Internal Medicine. I have become recertified in Internal Medicine two times.

Following completion of my residency training in Occupational Medicine, I was awarded a Fellowship in Occupational Medicine from the Foundation for Occupational Health and Research. I continued at Mount Sinai, where I joined the faculty, and continued to evaluate patients with asbestos exposure. I became Vice Chair of the Department of Preventive Medicine in 2001. I was Director of the New York/New Jersey Education and Research Center from 2006 – 2010, and had been Director of the Residency Program in Occupational Medicine from 1998-2006. I was also the Director of the Mount Sinai World Trade Center Medical Monitoring and Treatment Program from 2006 – 2010, although my involvement with the World Trade Center medical programs started in 2001, when I began to evaluate patients with exposure to the World Trade Center disaster, and was initially Medical Core Director of the World Trade Center Worker and Volunteer Medical Screening Program (2002-2004), and Co-Director of the World Trade Center Medical Monitoring and Treatment Program (2004-2006). I have published over fifty articles in the peer-reviewed literature.

In 2010, I left the Mount Sinai School of Medicine to become the Founding Chair of the Department of Population Health at Northwell Health and Hofstra Northwell School of Medicine (formerly known as North Shore University Health System). The Department changed its name in 2014 to Occupational Medicine, Epidemiology and Prevention.

I have evaluated hundreds of patients with asbestos exposure in my career in occupational medicine, spanning over 26 years. I currently direct the Occupational and Environmental Medicine Center of Long Island, providing occupational health services to patients in the metropolitan New York area. Over the past year alone, I have supervised the examination of or directly examined nearly 700 patients with asbestos exposure, as we have greatly expanded our clinical services. Over the course of the past 25 years, I have evaluated dozens of patients with malignant mesothelioma and lung cancer due to asbestos exposure. I have kept abreast of the scientific and medical literature regarding the diagnosis and causation of mesothelioma. I have personally evaluated cases of mesothelioma where the exposure was brief, and have also seen cases of mesothelioma in individuals whose only exposure to asbestos was from family members who worked with asbestos and brought their asbestos contaminated clothes home.

Materials Reviewed:

I have had the opportunity to review the medical records and deposition transcripts of Mr. Rimondi. I was provided with the following information:

1. Stony Brook University Hospital, medical records, pathology report, and radiology records.
2. Quest Medical, medical records dated August 22, 2016 to June 28, 2017
3. Family Medical Care, dated February 3, 2012 to November 22, 2016
4. ProHealth, medical and radiology records.
5. Plaintiff's Answers to standard interrogatories, dated August 8, 2017
6. Plaintiff's de bene esse deposition transcript sated August 23, 2017
7. Plaintiff's discovery deposition transcript, dated August 15, 2017
8. Discovery deposition transcript of plaintiff's aunt, Emma Pantle, dated October 12, 2017
9. Discovery deposition transcript of plaintiff's oldest son, Richard Gianfranco Rimondi , October 20, 2017
10. Discovery deposition transcript of plaintiff's wife, Pilar Rimondi, dated November 3, 2017
11. Investigation of Italian Talc by Steven P. Compton, PhD, dated August 1, 2017
12. Analysis of Johnson & Johnson Baby Powder and Valiant Shower to Shower Talc Products by William E. Longo, PhD, dated August 2, 2017
13. Below the waist application of Johnson & Johnson Baby Power report by William E. Longo, PhD, dated September 2017
14. Johnson & Johnson Baby Power use simulation report by Sean Fitzgerald, PG, dated September 14, 2017
15. Johnson & Johnson test report by Sean Fitzgerald, PG, dated July 26, 2017

Mr. Rimondi's Medical and Exposure History:

Clinical History: Mr. Rimondi is a 57 year old man who noted left sided flank swelling and discomfort in early July 2016, following a trip to Peru. He went to see his physician, Dr. Carla Weber, who ordered rib films. There was no evidence of fracture, and the visualized pleura appeared normal. The pain persisted, and was now present in the left thoracic area. Dr. Weber saw Mr. Rimondi on August 3, 2016, and there was mild tenderness on palpation. Dr. Weber ordered a CT scan. The CT scan was performed on August 12, 2016, and showed a moderate sized left pleural effusion with mild left lower lobe atelectasis. There was an abnormal soft tissue prominence/mass in the left posterior lower thorax, in the anterior left basilar pleural space and upper abdomen including the left retroperitoneum. An MRI with contrast was recommended.

Mr. Rimondi went to see Dr. Jose Rodriguez on August 22, 2016. He had lost around 13 pounds in three months. Dr. Rodriguez referred Mr. Rimondi to a thoracic surgeon. An MRI of the chest was performed on September 2, 2016, which showed an abnormal soft tissue mass that appeared extrathoracic at the level of the left posterior lateral

lower thorax and upper abdomen. It was not well distinguished from the peripheral musculature of the thorax and upper abdominal wall. There was an infiltrating mass extending into the pleural and retroperitoneal structures, extending into the perinephric fat. The mass also appeared to be present in the left anterior basilar pleural space and adjacent left anterior pericardial fat. The radiology noted that this finding was highly suspicious for malignancy. Dr. Rodriguez evaluated Mr. Rimondi on September 9, 2016. He was referred to thoracic oncology.

Mr. Rimondi had a left ultrasound-guided biopsy of the posterior chest wall/flank mass at Stony Brook on September 22, 2016. The cells were consistent with malignant mesothelioma, epithelioid cell type. Dr. Raja Flores saw Mr. Rimondi on October 6, 2016. Dr. Flores ordered a PET/CT scan, PFTs, and a MIBI scan. A PET scan was performed on October 21, 2016, which showed extensive malignant mesothelioma involving the left hemithorax and abdominal wall. As a result of these findings, Mr. Rimondi was not considered a surgical candidate and he was referred to oncology for chemotherapy. Dr. Jorge Gomez saw Mr. Rimondi and he started him on chemotherapy with Pemetrexed, Carboplatin, and Bevacizumab on November 21, 2016. Dr. Gomez also noted that Mr. Rimondi was being treated for a sore throat with antibiotics. Dr. Weber saw Mr. Rimondi on November 22, 2016. He had level 8/10 pain; he was prescribed Oxycodone. A brain MRI on November 22, 2016, showed no evidence of a mass, intracranial hemorrhage, or lobar infarct.

A CT scan was performed on December 22, 2016. The ill-defined mass like density within the left inferior aspect of the anterior mediastinal fat had not significantly changed. There was a slight decrease in the left-sided pleural effusion, but it remained moderate in size, with associated improvement in the aeration of the left lower lobe. Pericardial involvement adjacent to the lateral wall of the left ventricle could not be excluded. Scattered subcentimeter nodules were noted in both lungs. Mr. Rimondi went to the Emergency Department at Good Samaritan Hospital on December 30, 2016, with complaints of right upper quadrant abdominal pain. The pain was severe, radiating to his back and rates 10/10. Mr. Rimondi also had been vomiting for the past day. An abdominal ultrasound showed gallstones in the gallbladder neck with no evidence of biliary dilatation. A chest x-ray showed no active pulmonary disease. A CT scan of the abdomen and pelvis showed non-specific distention of the gallbladder. There was a partially imaged left pleural effusion. There was pleural thickening at the left posterior costophrenic angle and along the left hemidiaphragm. Non-specific soft tissue thickening was seen along the left lateral abdominal wall. Mr. Rimondi had an elevated lactate level.

He underwent surgery for a gangrenous gallbladder and had an open cholecystectomy. Dr. John Hsu performed the surgery. At a follow-up visit on January 12, 2017, Dr. Hsu noted that Mr. Rimondi was having residual wound issues related to wound healing from a grossly infected surgical field and like related to the effects of chemotherapy on wound healing. Dr. Hsu planned to talk to Dr. Gomez about delaying chemotherapy to allow for wound healing. Mr. Rimondi returned to Dr. Hsu on January 24, 2017. There were still two spots open with minimal drainage, but the wound was healing. Dr. Hsu felt that it would be appropriate to restart chemotherapy. At a return visit on February 8th, Dr.

Hsu noted that the open cholecystectomy wound was closed and that Mr. Rimondi was doing well from a surgical standpoint.

Dr. Hsu evaluated Mr. Rimondi on March 2, 2017. He appeared pale and fatigued and had just received his fifth cycle of chemotherapy. His wounds had healed completely. Dr. Rodriguez saw Mr. Rimondi on March 29, 2017. Mr. Rimondi was now on insulin for diabetes that was diagnosed during chemotherapy treatment. Dr. Francisco Garcia-Moreno, a medical oncologist, saw Mr. Rimondi on March 31, 2017. He noted that Mr. Rimondi had completed six cycles of Pemetrexed, Carboplatin, and Bevacizumab with a good response. He was now to receive maintenance Bevacizumab, and requested a transfer to an oncologist closer to home. At the time of the visit, Mr. Rimondi had night sweats, fatigue, weakness, insomnia, and weight loss. He also had trouble with his vision, post nasal drip and a distortion in taste. Mr. Rimondi was anxious and depressed. He had headaches, dizziness, numbness and tingling. He had lost an additional 15 pounds since the summer. Dr. Garcia-Moreno discussed that the treatment was palliative and that the mesothelioma was incurable. The single agent Bevacizumab started on April 11, 2017.

Mr. Rimondi returned to Dr. Garcia-Moreno on April 25, 2017. He noted soaking night sweats on the Bevacizumab. Dr. Garcia-Moreno felt that night sweats were likely related to the underlying malignancy, and noted that they had been occurring since the fourth dose of chemotherapy. Dr. Garcia-Moreno planned to monitor him, and he was to receive the next cycle of Bevacizumab on May 2, 2017. He tolerated the Bevacizumab without side effects. He received his third cycle on May 23, 2017, and fourth cycle on June 13th, 2017. Dr. Garcia-Moreno saw Mr. Rimondi on June 23, 2017. He was stable and had received the four cycles of Bevacizumab. Mr. Rimondi was to get a repeat CT scan for staging. Mr. Rimondi was no longer taking insulin with the discontinuation of the steroids. His fifth cycle of Bevacizumab was administered on July 5th. A CT scan on July 7, 2017, showed thickening of the left pleura and the upper left lateral abdominal wall musculature. There were several subcentimeter bilateral pulmonary nodules. The pleural thickening had decreased minimally since March, but the abdominal wall musculature thickening was unchanged. Dr. Garcia-Moreno saw Mr. Rimondi on July 12, 2017. He noted that the reports from the CT scan showed stable disease. He received his sixth dose of Bevacizumab on July 26th. He noted occasional facial flushing.

A CT scan on August 24, 2017, showed increased pleural thickening near the diaphragm, in the posterior upper pleura, along the left heart border, and new thickening of the posterior pleura near the left costophrenic angle. There was an increase in the thickening of the soft tissues in the left upper abdominal/lateral chest wall musculature. No additional medical records were provided. Dr. David Zhang had the opportunity to meet with Mr. Rimondi on January 10, 2018. Mr. Rimondi stated that the Bevacizumab was stopped in August and he started Keytrude in December 2017. At the time of Dr. Zhang's evaluation, Mr. Rimondi complained of bone pain and pulsating pain in the left chest. He had generalized weakness, night sweats, loss of appetite, weight loss and depression. Mr. Rimondi also noted dizziness, numbness, abdominal cramps and diarrhea, along with insomnia.

Past Medical History: Mr. Rimondi has a history of gastroesophageal reflux disease. He has degenerative disc disease, carpal tunnel syndrome, and has undergone cervical spine surgery and hip surgery. He has a history of irritable bowel syndrome, hypercholesterolemia, fatty liver and gallstones.

Cigarette Smoking History: Mr. Rimondi smoked around a half pack of cigarettes a day for 10 years, giving him an approximate 5 pack-year smoking history.

Environmental and Occupational History: Mr. Rimondi grew up in Peru and moved to the United States in 1992. He worked as an accounting assistant and in the accounting department for the police department in Peru. When he came to the US, he worked packing cosmetic products from 1995 -1996 and then packed products for a 99¢ story from 1996 – 1997. He worked for United Pet Group from 1997-1998 weighing additives for use in pet food. He then moved to Entenmann's, a baked goods manufacturer, where he worked from 1998 until 2010 when he was disabled. He is unaware of any occupational exposure to asbestos.

Mr. Rimondi used Johnson's Baby Powder (JBP) from when he was an infant. His mother or aunt applied it to him when he was a baby. He recalled that his mother used JBP on his whole body after she bathed him. His aunt, Ms. Emma Pantile, noted that she applied JBP to Mr. Rimondi when she changed his diaper and after his bath; he lived with his aunt during the week, and with his mother on the weekends. She applied the JBP with a powder puff. Mr. Rimondi began applying the powder himself when he was around 6 years old. Ms. Pantile noted that he would come out of the bathroom after applying the powder and "come out all white because of the powder or the talc." He continued this practice with his children, and continued to personally use the JBP twice a day. He applied it to his body below his neck, including his chest, underarms and "private parts." He poured the powder into his hands and applied it to his arms and legs and feet. He used two to three containers of talcum powder per month. Mr. Rimondi applied JBP to his sons after they were born. He put powder on at each diaper change and after he bathed them. He estimated that they were in diapers until about three years old, and he changed their diapers about two to three times a day. Mr. Rimondi noted that the powder would become "like a cloud" when he applied it to his body, and he could see excess powder on the dresser and the floor. He would clean the floor, or his wife cleaned the floor. Powder would be present on the bedroom dresser after he used the powder. Mr. Rimondi applied the JBP in the bathroom after he moved to the United States. He stopped using the powder when he stopped working in 2010.

Conclusion: Mr. Rimondi has malignant mesothelioma of the pleura as a result of his cumulative exposure to talcum powder exposure. He has undergone chemotherapy and treatments. Based on the information available, it is my opinion, to a reasonable degree of medical certainty that Mr. Rimondi's exposure to asbestos-containing talcum powder led to the development of her mesothelioma. His mother and aunt used talcum powder on him as a child, and he began using talcum powders himself as a child, and continued using powders for the next 50+ years. Besides applying talcum powder to his own body, she used talcum powder on his children, during diaper changes, and after their baths for many years.

The methodology and basis for my opinions follows standard methods of the medical and scientific community. Asbestos is the most well known cause of mesothelioma, and the causation of mesothelioma has been established by the quantitative history of exposure to asbestos. Thousands of individuals, from myriad professions and exposure situations have developed mesothelioma as a result of either direct or indirect exposure to asbestos. The reliance on the history of exposure to asbestos was used by seminal studies by Newhouse, Wagner and Selikoff in the 1960s, who attributed mesothelioma to asbestos exposure based solely on the history of exposure. The increased risks for mesothelioma exist for individuals who both worked directly with asbestos products and for those who worked adjacent to or in the vicinity of others who were using asbestos products, which is known as "bystander" exposure.

Asbestos and Malignant Mesothelioma General Opinions: Occupational Medicine is the field of medicine that deals with exposures to substances, toxins, conditions and agents in the workplace that are associated with increased risks of diseases. It exists as a subspecialty of Preventive Medicine that deals with identifying ways to prevent people from becoming ill. This includes identifying the sources, agents or catalysts that increase the likelihood of someone developing a disease, illness, or detrimental condition, and educating people on how to eliminate, avoid, and/or mitigate those risks. To put it simply, Occupational Medicine and Preventive Medicine involves searching for and identifying causes of diseases. This knowledge is important for those who are already ill: elimination of the catalysts can eliminate or mitigate the illness. It is also important from a public health point of view: to a large extent, the higher purpose of Occupational Medicine and Preventive Medicine is to educate and warn the public on how to eliminate, avoid, or mitigate the risks of diseases at the workplace, and to provide guidance to governments and businesses on appropriate regulations and standards concerning workplace health and safety.

One of the essential tasks of a physician of Occupational Medicine, when dealing with an individual patient, is the taking of a proper occupational history. Standard medical histories usually involve the patient explaining their reason for seeking medical attention; a listing of current symptoms, conditions, allergies, medications and other relevant medical problems; and providing some family and social history. Occasionally, a standard medical history may-but doesn't always-include identifying the patient's occupation.

A full occupational history, on the other hand, will go into details of a patient's entire work history, including details concerning their tasks and duties and their working conditions and environment. The history will also routinely make inquiries into the patient's home or hobbies. It would also reveal what kinds of substances or agents the patient was exposed to in his or her working environment that might have occurred decades earlier. It remains the standard tool for determining exposure and has not been supplanted by quantitative measurements, which are rarely obtained, and would not, unless continuously performed on an individual (which is not feasible), fully address all exposures an individual might have had. At times, it is not possible to directly obtain an occupational history from an individual, and information concerning work and environmental experiences contained in deposition transcripts by plaintiffs, co-workers and family

members can provide detailed information of that type that can be elicited from an occupational physician-obtained history.

The hallmark of occupational medicine is to connect an exposure to a hazardous substance to a disease, and identify whether there is a causal relationship. This is a critical differentiation in the field of occupational medicine; not only do we treat patients for disease, but we emphasize what hazardous substance might be causing the disease. In occupational medicine training, there are core areas of training, including epidemiology, biostatistics, toxicology, and industrial hygiene.

Asbestos and Disease: Asbestos is a naturally occurring mineral that has been used commercially for a variety of purposes for over 100 years. Asbestos is mined in the form of microscopic fibers released from the surrounding earth. Asbestos was extremely useful from an industrial perspective: it is highly resistant to heat and therefore serves as an excellent insulator and friction surface. It is also very durable, and as a fiber it can be molded into shapes and products that serve a variety of functions. However, asbestos is also highly toxic and carcinogenic when the fibers are inhaled or ingested.

While there are many "fiber types" of asbestos, as well as different sizes of the fibers, there exists consensus among scientists that exposure to *any* asbestos fiber type or size increases the likelihood of lung cancer, mesothelioma, as well as nonmalignant lung and pleural disorders. Asbestos fibers are generally divided into two categories: amphiboles and serpentine (or chrysotile). There are several varieties of amphiboles, including both commercial and non-commercial types. The three major asbestos types used in industry have been chrysotile, amosite and crocidolite. Of these three fiber types, over 95% of all asbestos used in the United States has been chrysotile. Much of the chrysotile asbestos that was used in the US was mined in Canada, where there was contamination with small amounts of tremolite, another type of amphibole asbestos. The mainstream scientific community has also long recognized, and continues to recognize today, that there is no "safe" level of exposure to asbestos regardless of fiber type or size. This position is shared by numerous United States government agencies, including the Occupational Safety and Health Administration ("OSHA", which has regulatory authority over workplaces), the Environmental Protection Agency ("EPA" which has regulatory authority over non-occupational settings), the National Institute for Occupational Safety and Health ("NIOSH", which is responsible for conducting research and making recommendations for the prevention of work-related injuries and illnesses), the World Trade Organization ("WTO"), and the national academies of science of every major industrialized nation. The World Health Organization recently reviewed the existing literature and concluded (in 2014) that all fiber types are capable of causing asbestos related disease, including mesothelioma, and reiterated the statement that there is no safe level for exposure to asbestos.

Due to the ubiquitous use of asbestos and its presence in naturally occurring formations, there is asbestos in the ambient air in the United States, albeit at minute levels. The ambient air concentration or "background level" has been reported to range from 0.0005 f/cc in urban areas, to 0.00005 f/cc in rural regions. These levels are thousands of

times less than the current OSHA permissible exposure level of 0.1 f/cc. While it is theoretically possible to develop mesothelioma from ambient air concentrations, it has not been proven to occur at levels at or below ambient air concentrations. Given that there is no truly "unexposed" population, it would be impossible to reasonably perform such a study to determine if this were the case.

State of the Art:

In 1898, Montague Murray described interstitial fibrosis in an individual exposed to asbestos. Pancoast described radiographic changes of interstitial fibrosis in asbestos workers in 1917. Cooke described two cases of asbestosis in the 1920s, and actually used the term "asbestosis" to describe the interstitial fibrosis among asbestos workers, and also noted pleural plaques (fibrosis) in these workers.

In 1930, Merewether and Price, in their *Report on the Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry,* noted that inhaling dust containing asbestos fibers could lead to disabling and fatal lung disease. They studied asbestos workers in the textile mills in Great Britain, and noted that asbestosis could occur in large numbers of exposed individuals. Moreover, they found that the textile workers with the highest exposures had more asbestosis than workers in areas where asbestos exposure was lower. Merewether and Price noted that asbestos was a potential hazard to health in any industry where dry asbestos products were abraded or otherwise manipulated to generate dust, such as thermal insulating. They recommended warning, education and training of all those individuals who were exposed to asbestos.

Lynch and Smith noted a case of lung cancer in an asbestos worker from South Carolina in 1935. Textbooks in the 1930s, such as A.J. Lanza's textbook on dust disease, included asbestosis as a disease of concern. In 1943, the first case of mesothelioma was associated with asbestos exposure and was published by Wedler in Germany. Also in 1943, Hueper from the United States Public Health Service stated that he believed asbestos caused lung cancer. He published an editorial stating this association in the Journal of the American Medical Association in 1949.

In 1955, Doll published a seminal article that described the increased risk of lung cancer among asbestos exposed workers. By the time of Doll's epidemiology study, there had been over 60 cases of asbestos-related lung cancer published in the literature. In 1960, Wagner et.al. published a study of 33 cases of malignant mesothelioma among individuals who were exposed to asbestos in and around the crocidolite mines in South Africa. Not only were miners developing disease, but family members, individuals on the wagon routes in which the asbestos was carried and people who had played with mine tailings as children developed mesothelioma. In the early 1960s numerous studies in several countries, under different exposure scenarios, were published that showed mesothelioma in association with asbestos exposure. In fact, by the end of 1964, over 700 scientific articles had been published that showed the adverse health effects of asbestos.

The Development of Diseases: When asbestos is inhaled, some proportion of the fibers can be deposited upon any component of the respiratory tract, including the nose, pharynx, conducting airways and the alveolar or gas exchanging regions of the lung. Fibers that land initially on the airways and above are cleared rapidly from the lung. The primary defense mechanism that mediated this clearance is known as the mucociliary escalator. The escalator is comprised of collated and mucous producing epithelial cells that propel inhaled fibers up to the mouth where they can be swallowed or expectorated. These epithelial lining cells are the "target cells" for cancers. Fibers that evade the mucociliary escalator can penetrate into the lower airways and lung tissue, where they can be transported through the body. Amphibole fibers tend to clear from the lung less rapidly than chrysotile fibers. Asbestos is cleared through the pulmonary lymphatics to lymph nodes and to the pleura, the target organ for pleural mesothelioma. Of the different fiber types, Suzuki, Sebastien and LeBouffant have all shown that chrysotile fibers preferentially translocate to the pleural space.

Asbestosis: The fibers that are inhaled and deposited past the escalator can cause asbestosis. These fibers deposit initially on the Type 1 and Type 2 alveolar epithelial cells. On the epithelial surfaces, some asbestos fibers activate the 5th complement which attracts inflammatory cells, including foreign particles, like asbestos, from the lung. About 20% of the fibers deposited on the alveolar surfaces are enveloped by the Type 1 cells and are translocated to the underlying connective tissue (interstitial) compartment. There, the fibers can interact with interstitial fibroblasts, myofibroblasts and macrophages. Fibroblasts and myofibroblasts are the target cells for asbestos because these are the cells that synthesize and release the scar tissue matrix. (See Y. Suzuki & N. Kohyama, *Translocation of Inhaled Asbestos Fibers from the Lung to Other Tissues.,*19 Am J. Indus, Med. 701 (1990); Y. Suzuki & N. Kohyama, *Translocation of Inhaled Asbestos Fibers from the Lung to Other Tissues*., *19* Am J. Indus, Med. 701 (1991)). They produce scar tissue when the epithelial cells are injured and when the macrophages are activated. Alveolar cells and macrophages release a number of protein growth factors that stimulate the fibroblasts to multiply and produce scar tissue and the fibroblasts and myofibroblasts also synthesize a similar array of factors that induce their own cell growth and matrix production that we recognize as asbestosis. Like *all* of the asbestos-related diseases, asbestosis is dose dependent. An individual typically needs long-term occupational exposure to develop clinical asbestosis.

The scarring process described above begins as soon as inhaled fibers are deposited on the alveolar surfaces, and microscopic asbestosis is ongoing in the lungs of afflicted individuals for many years before any clinical signs or symptoms are presented. The initial physiological symptom of asbestosis is shortness of breath. This is caused by the scar tissue which replaces normal elastic connective tissue, this producing a stiff lung that restricts the individual from taking a deep breath. Shortness of breath also results when scar tissue thickens the alveolar-capillary membrane, the barrier across which oxygen and carbon dioxide gases are exchanged.

Pleural Plaques and Fibrosis: This is scar tissue formation in an identical manner to that described above, under asbestosis. The difference is that there is little direct deposition of asbestos fibers in the pleura. While some fibers can be inhaled through the alveolar ducts

and reach the pleura directly, most fibers that land on alveolar surfaces and reach the interstitial compartment have direct access to the pleura do so by way of pulmonary lymphatic flow. The inhaled fibers that land on alveolar surfaces and reach the interstitial compartment have direct access to lymphatic fluids which flow through these regions on the way to the pleura. The lymphatic flow carries fibers to the pleura where they interact with the sub-mesothelial fibroblasts that produce a scar tissue matrix, as described above. If the scarring is in a circumscribed pattern, the scarring is called "plaque". Investigators have shown that this injury can result in a restrictive lung disease in some individuals.

Lung Cancer: These tumors caused by asbestos typically arise in cigarette smokers, although some epidemiologic studies on asbestos-exposed non-smokers show an increased risk of developing the disease. When an individual is exposed to the cancer-causing agents (carcinogens) of both cigarettes and asbestos, the risk of getting lung cancer is increased well beyond the risk presented by exposure to either agent alone or by simply adding the risks of the two carcinogens. Epidemiologists multiply the risks of the two carcinogens since there is a clear synergy in the way asbestos and cigarette smoke combine to cause lung cancer.

Cancer is the loss of control of cell growth. Every cell in the bodies of humans and animals is under strict genetic control of the rate at which a given cell replaces itself by dividing. Cancer is caused when the specific genes that control cell division and other aspects of the cell cycle develop errors or mutations. Carcinogens induce such errors, and complete carcinogens can produce the errors with no other agent required. Cigarette smoke has a number of complete carcinogens, and all of the asbestos varieties have been shown to act as complete carcinogens. Thus, as the airway epithelial cells of the mucociliary escalator are assaulted daily by cigarette smoke and asbestos fibers, a number of cells are injured, and many exhibit genetic errors through the lifespan of the individual. In those who are susceptible to developing a cancer, one of those injured cells accumulates a sufficient number of genetic errors in genes that control cell growth to finally, after decades of exposure, lose the normal growth pattern and grow into a malignant tumor. (See Frost G, Darton A, Harding AH. *The effect of smoking on the risk of lung cancer mortality for asbestos workers in Great Britain (1971-2005)* Ann Occup Hyg 55:239-24 (2011)).

Mesothelioma: This cancer occurs when a mesothelial cell of the pleural or peritoneal surfaces develops a sufficient number of genetic errors in a set of genes that control cell growth, as described above. Cigarette smoking has no influence on the development of mesothelioma. (See N.S. Offermans, et. al., *Occupational Asbestos Exposure and Risk of Pleural Mesothelioma, Lung Cancer, and Laryngeal Cancer in the Prospective Netherland Cohort Study,* 56 J. Occupational Envt'l Med. 1 (2014); Robinson BM. *Malignant pleural mesothelioma: an epidemiological perspective,* 1 Annals Cardiothoracic Surgery 491 (2012)).

Asbestos exposure is the only known occupational and/or environmental cause of mesothelioma in North America, and all of the asbestos varieties induce the genetic errors described above and cause this cancer. The fibers that cause mesothelioma reach the pleural surfaces through the lymphatic pathways, as explained earlier, and they interact with the

target cells of the mesothelial surfaces. When a sufficient number of genetic errors have accumulated in a single mesothelial cell, this cell can undergo neoplastic transformation and grow into a deadly tumor. It typically takes many decades for a sufficient number of mutations to occur in a single mesothelial cell because of the numerous effective defense mechanisms that destroy genetically defective cells, thus explaining the long latencies known for this cancer.

All of the asbestos varieties have been shown to cause genetic errors and fibers less than five microns can bind DNA and this contributes to the development of genetic damage. Short fibers have been found to accumulate in the pleural regions of the lung as well as in mesenteric lymph nodes of the peritoneal cavity. Longer fibers may be comparatively more dangerous than short fibers (on a fiber per fiber basis), but all size ranges are capable of causing and contributing to the development of mesothelioma or any of the asbestos-related diseases. Exposure to asbestos fibers of all types and lengths are toxic, and short fibers more readily reach the mesothelial target cells of the pleura. (See Y. Suzuki & S. R. Yeun, *Asbestos Fibers Contributing to the Induction of Human malignant mesothelioma.,* 982 Annals N.Y. Acad. Sci. (2002); Y. Suzuki, et al. *Short thin asbestos fibers contribute to the development of human malignant mesothelioma: pathological evidence.,* 208 Int'l. J. Hygiene Env. Health 201 (2005)). Some have suggested that geological nomenclature – calling the anthophyllite and tremolite in the talc either "non-asbestiform" or "cleavage fragments" – has biological significance. This notion has been rejected by the EPA, US Centers for Disease Control and Prevention, Agency for Toxic Substances and Disease Registry, and American Thoracic Society, and is not a distinction that is considered medically important. In fact, mesotheliomas have been documented among New York State miners and millers of talc containing approximately 50% "non-asbestiform" anthophyllite and tremolite. Asbestos related diseases have also been found at the Vermont talc mines and mills. The absence of documented cases of mesothelioma among one cohort of miners and millers of talc containing less than 1% the tremolite and anthophyllite (such as the Italian studies of talc miners and millers) is most likely due to an inadequate sample size. (US EPA Region 9 Response to the 2005 National Stone, Sand and Gravel Association Report, April 20, 2006; RT Vanderbilt Co., MSDS, May 1, 1975; Roggli, et.al. *Tremolite and Mesothelioma.,* Ann Occ Hyg 46(5):447-453 (2002); Lamm, *Similarities in Lung Cancer and Respiratory Disease Mortality of Vermont and New York State Talc Workers;* Epidemiology-Fibers, 1576-1581 (1988)).

Fibers of all lengths can bind to DNA and cause genetic errors that are required in the causation of cancer such as mesothelioma. Fiber burden studies of mesothelioma patients show a preponderance of chrysotile asbestos within the tumor tissue. Since the target location of mesothelioma is the pleura, the lung burden of asbestos does not reflect the fact that asbestos has moved from the lung to the pleura, where it can cause the mesothelioma to develop. (See Ronald F. Dodson, *Analysis and Relevance of Asbestos Burden in Tissue, in Asbestos: Risk Assessment, Epidemiology and Health Effects.* Risk Assessment, Epidemiology and Health Effects 78 (2d, ed. 2011); M. Silverstein, et al., *Developments in Asbestos Cancer Risk Assessment.* Am J. of Indus. Med. (2009)).

Moreover, there is ample evidence to support the conclusion that exposure to the asbestos fibers typically used in brake linings-chrysotile fibers-can and does cause mesothelioma. This conclusion is supported by, among others, the American Conference of Governmental Industrial Hygienists, the American Thoracic Society, the Environmental Protection Agency, the International Agency for Research on Cancer, the National Toxicology Program, OSHA, the Consumer Products Safety Commission, the World Health Organization, and the World Trade Organization. The scientific consensus that all fiber types and sizes can cause mesothelioma is also reflected in the Consensus Report of the 1997 Helsinki Conference (discussed below) and publications from the American Cancer Society and the National Cancer Institute of the National Institutes of Health.

In essence, there exists a consensus among the overwhelming majority of medical and scientific professionals and organizations that asbestos fibers of any type or size can cause mesothelioma, including chrysotile fibers. (See Dodson, Ronald F. et al., *Asbestos Fiber Length as Related to Potential Pathogenicity: A Critical Review,* 44 Am J. Indus. Med. 291 (2003); D. Egilman, et al., *Exposing the "Myth" of ABC, "Anything But Chrysotile: A Critique of the Canadian Asbestos Mining Industry and McGill University Chrysotile Studies.* 44 Am J. Indus. Med. 540 (2003); David S. Egilman & Marion Billings: *Abuse of Epidemiology: Automobile Manufacturers Manufacture a Defense to Asbestos Liability,* 11 Int. J. Occupational Envtl Health 360 (2005). 11:360-371; Egilman D. *Fiber Types, Asbestos Potency, and Environmental Causation.* 15 Int. J. Occupational Envtl. Health (2009); Finkelstein, M. *Asbestos Fiber Concentrations in the Lungs of Brake Workers: Another Look,* 52 Annals Occupational Hygiene 455 (2008); M.M. Finkelstein & C. Meisenkothen, *Malignant Mesothelioma among Employees of a Connecticut Factory that Manufactured Friction Materials Using Chrysotile Asbestos.* 54 Annals Occupational Hygiene 692 (2010); P.J. Landrigan, et al., *The Hazards of Chrysotile Asbestos, a Critical Review.* 37 Indus. Health 271 (1999); W.J. Nicholson, *The Carcinogenicity of Chrysotile Asbestos-A Review.* 39 Indus. Health 57 (2001); R.A. Lemen, *Chrysotile Asbestos as a Cause of Mesothelioma: Application of the Hill Causation Model.* 10 (2) Int. J. Occupational Envtl. Health (2004); *see also* R. Lemen, *Asbestos in Brakes: Exposure and Risk of Disease.* 45 Am. J. Indus. Med 229 (2004); EPA: *Guidance For Preventing Asbestos Disease Among Auto Mechanics.* (1986); A.H. Smith & C.C. Wright, *Chrysotile Asbestos is the Main Cause of Pleural Mesothelioma.* 30 Am. J. Indus. Med. 252 (1996); U.S. Dept. of Labor: *Working Safely with Asbestos in Clutch and Brake Linings.* (posting); U.S. Dept. of Labor, OSHA Directorate of Science, Technology and Medicine, Office of Science and Technology Assessment. *Asbestos-Automotive Brake and Clutch Repair Work*; World Health Organization, *Environmental Health Criteria 203: Chrysotile Asbestos.* International Programme on Chemical Safety (1998 Geneva)).

Asbestos fibers are very small; so small, in fact, that millions of fibers could fill the air in a room without anyone being able to perceive it with the naked eye. The fibers are odorless, cannot be seen with the naked eye, and are aerodynamic. Consequently, someone can inhale asbestos fibers without even being aware of it. The fibers are also small enough to pass through the normal respiratory defense mechanisms that the human body uses to keep out toxins and debris.

The Scientific community has even concluded that small amount of asbestos exposure can cause cancer. The Rodelsperger study indicates that exposure to asbestos below the Occupational Safety and Health Administration (OSHA) Permissible Exposure Level (PEL) of 0.1 fibers per cubic centimeter can cause disease. However, visible asbestos-laden dust that is released into the air from the manipulation of gaskets or packing, or that is reintroduced into the respirable zone from the process of sweeping the floor, is between 2.0 and 10.0 fibers per cubic centimeter. These levels far exceed the OSHA PEL. Some of these levels even exceed the OSHA PEL issued in 1972.

Government agencies and international organizations universally recognize asbestos as a carcinogen in low levels. These agencies include the International Agency for Research on Cancer, Environmental Protection Agency, OSHA, National Institute for Occupational Safety and Health, and World Health Organization. The inhalation of asbestos fibers also does not trigger any immediate physiological reactions: the victim doesn't experience any immediate irritation, asthmatic problems, or allergic reactions. Moreover, the latency, or development period, for mesothelioma is very long: the minimum latency period is usually considered to be around 10 years with a maximal latency period well over 60 years after the last exposure. Consequently, it could be decades before someone is aware that he or she was exposed to asbestos, or it might have occurred so remotely that they do not realize they had asbestos exposure. Moreover, they may not realize that a product they used contained asbestos and thus are unaware they had exposure.

The Helsinki Criteria for Attribution: In January 1997, a conference called "Asbestos, Asbestosis and Cancer" was held in Helsinki, Finland. The conference was convened to establish criteria for diagnosis and attribution of disorders of the lungs and pleura, including mesothelioma. This was a multidisciplinary group of internationally recognized experts, consisting of pathologists, radiologists, occupational and pulmonary physicians, epidemiologists, toxicologists, industrial hygienists, and clinical and laboratory scientists specializing in tissue fiber analysis. Collectively, the members had published over 1,000 articles on asbestos and associated disorders. The conclusions of the conference were developed into a peer-reviewed Consensus Report that established the "Helsinki Criterion". Among the conclusions of the Helsinki Criterion are:

a. That, in general, reliable work histories provide the most practical and useful measures of occupational asbestos exposure; and
b. That even in the absence of other independent evidence of disease (e.g. lung fiber counts exceeding the background range for the lab in question; the presence of radiographic or pathological evidence of asbestos-related tissue injury; histopathologic evidence of abnormal asbestos content), a history of significant occupational, domestic or environmental exposure to asbestos will suffice for attribution of the disease with asbestos exposure.

Moreover, with reference to determining an occupational etiology of mesothelioma, the Helsinki Criterion Consensus Report concluded that:

a. The great majority of mesotheliomas are due to asbestos exposure;

b. Mesothelioma can occur in cases with low asbestos exposures. However, very low background environmental exposures carry only an extremely low risk;
c. About 80% of mesothelioma patients have had some sort of occupational exposure to asbestos (necessitating a carefully obtained and detailed occupational history for proper diagnosis);
d. An occupational history of brief or low-level exposure should be considered sufficient for mesothelioma to be designated as occupationally related;
e. A minimum of 10 years from the first exposure is required to attribute mesothelioma to asbestos exposure (though in most cases, the latency interval is longer);
f. Smoking has no influence on the risk of mesothelioma.

The conclusions of the Helsinki Criterion have since been adopted by, and form the general consensus of, the medical community's positions vis-à-vis mesothelioma and asbestos. (See *Consensus Report, Asbestos, asbestosis and cancer: the Helsinki criteria for diagnosis and attribution,* 23 Scandinavian J. Work Environ Health 311 (1997)). And, given the fact that about 80% of patients with mesothelioma have had some sort of occupational exposure to asbestos,[1] asbestos exposure in the workplace is a prime focus of Occupational Medicine when dealing with mesothelioma patients.

Mesothelioma is a dose responsive disease: It is my opinion that Mesothelioma and asbestos related lung cancer are dose responsive diseases in which more substantial exposures directly increases the risk for the development of these cancers. This linear dose-response relationship presented in *Asbestiform Fibers: Non-occupational Health Risks,* published by the National Research Council National Academy of Sciences in 1984, discussed herein, is neither new nor novel and generally accepted in the medical and scientific communities. As per the aforementioned Helsinki criteria, the first question usually asked of a patient diagnosed with mesothelioma, concerns how, when, and where the patient was exposed to asbestos. (*See Consensus Report, Asbestos asbestosis and cancer: The Helsinki criteria for diagnosis and attribution.* 23 Scandinavian J. Work Environ Health 311 (1997)). Because of the proven association between asbestos fibers and mesothelioma, proof of significant exposure to asbestos dust is considered to be proof of specific causation. (*See* P. Boffetta, et al., *Health Effects of Asbestos Exposure in Humans: A Quantitative Assessment.* 89 (6) Medicina Del Lavoro, 471 (1998). This causal relationship between exposure to asbestos dust and the development of mesothelioma is so firmly established in the scientific literature that it is accepted as a scientific "fact".

Malignant mesothelioma is, in general, a dose response disease where all significant exposure to asbestos-containing dust has been shown to contribute to cause diffuse malignant mesothelioma including pleural mesothelioma (*See also* Newman, et al., *Malignant Mesothelioma Register 1987-1999.* 74 Int'l Arch Env. Health 383 (2001),

[1] The remaining 20% of mesothelioma patients likely had asbestos exposures that were para-occupational or are simply unidentified.

(concluding that "higher cumulative asbestos-fiber dose leads to the earlier development of mesothelioma)). As each exposure to asbestos contributes to the total amount of asbestos that is inhaled, and, in doing so, reduces the necessary period for asbestos disease to develop. Therefore, each non-trivial exposure to asbestos should be considered a substantial contributing factor in the development of the malignant mesothelioma or lung cancer.

Exposure to Asbestos contaminated talc and disease: Asbestos fibers have been reported in cosmetic talcum powder for decades, in company documents, the media, FDA communications, and the published medical and scientific literature. In 1935 asbestos was identified as a source of exposure in talc miners and millers by Dreesen. By 1968 Cralley had described asbestos in consumer cosmetic talc products. By 1972, the cosmetic industry was looking for asbestos free alternatives to cosmetic talc. Cosmetic talc has been analyzed by researchers in various countries, and has routinely been shown to be contaminated with asbestos. Exposure to asbestos contaminated talc has been shown to cause asbestos related diseases, including mesothelioma. In 1974 Rohl, and in 1976, Rohl and Langer tested 20 consumer products that had been labeled as talc or talcum powder, including body powders. Of the 20 products that were tested, ten were found to contain tremolite and anthophyllite, principally asbestiform. Of note, the product that had the highest asbestos content in the Rohl and Langer study was the same product later tested by Gordon, et.al. Mattenklott et. al. in 2007 found that small amounts of talcum powder (0.1 gram) released thousands of asbestos fibers. A recent paper by Gordon, et.al., Asbestos in Commercial Cosmetic Talcum Powder as a Cause of Mesothelioma in Women, evaluated the mineralogical constituents of Cashmere Bouquet and its ability to release asbestos fibers into the breathing zone of the direct user and bystanders. In their paper Gordon et.al. noted that the talc that was used in Cashmere Bouquet was derived from three distinct regions, where anthophyllite and tremolite asbestos were found, regions from which the talc used in Johnson's Baby Powder was also sourced. Gordon et.al. measured 18 million anthophyllite asbestos fibers per gram in the talcum powder. Air measurements were done by both phase contrast microscopy (PCM) and transmission electron microscopy (TEM), and significant levels of asbestos fibers were noted (anthophyllite, tremolite and some chrysotile) in the breathing zone of the individual applying the powder as well as a bystander. Results taken from the experiment in the paper show that personal measurements from the shaker container test showed a measurement by PCM of 4.8 f/cc, with an actual asbestos fiber measurement of 1.8 f/cc. Bystander measurements showed a lower, but still significant exposure of 1.35 f/cc by PCM for the bystander, and 0.5 f/cc of actual asbestos fibers. Similar measurements were done with the puff application method. Personal measurements after using a puff were 23.6 f/cc and 16.5 f/cc for the user, with actual asbestos fiber measurements of 5 f/cc and 3.5 f/cc. A short term sample showed even higher measurements, of 60 f/cc with the use of a puff and actual asbestos fiber measurements of 13 f/cc. Bystander exposures to asbestos from the puff application were elevated, with a short term sample by PCM of 13.7 f/cc and 9.7 f/cc, and an actual asbestos fiber measurement of 4.9 f/cc and 3.5 f/cc. Gordon et.al. also noted that the TEM measurements were far more sensitive than x-ray diffraction detection, since there was a much lower detection limit with TEM. In addition, the Mine Safety and Health Administration (MSHA) monitored personnel in the mill where Italian talc was ground (this talc was used in

consumer products) in 1984. The filters form the personal measurements from these workers contained 5.8% anthophyllite. The MSHA scientist determined that this equated to anthophyllite comprising 0.6% of the bulk Italian talc.

In addition to looking at bulk and air samples, Gordon et.al analyzed the lung tissue and lymph node tissue of a woman who had been exposed to contaminated talcum powder (Cashmere Bouquet). The authors found that there were 3150 and 4150 fibers per gram wet weight, respectively, with a detection limit of 690 fibers per gram wet weight. All fibers were 5 micrometers or greater in length, and had an aspect ratio of 20:1 or greater. The fibers were identified as anthophyllite or tremolite. In addition to the fibers counted above, there were many anthophyllite and tremolite fibers that were less than 5 micrometers in length, with a predominance of anthophyllite. In the lymph node, amphibole asbestos fibers were also noted, measuring 12,738 fibers per gram wet weight (detection limit 2123 fibers per gram wet weight). Again, the fibers noted were anthophyllite and tremolite. In addition to the asbestos found in the lungs, the authors noted fibrous and platy talc and small asbestos bodies.

The issue of asbestos and talc has been studied for decades. Millman in 1941 noted pneumoconiosis in a man exposed to cosmetic talc. Lung scarring was seen in miners from New York State in the 1950s, and there are elevated rates of mesothelioma and lung cancer in miners at the asbestos contaminated talc mines. The International Agency for Research on Cancer has noted that talc contaminated with asbestos is carcinogenic.

Applying an Accepted Method for Evaluating Disease Causation in an Individual:
In deciding whether Mr. Rimondi's mesothelioma was caused by her exposure to asbestos, I applied the methodology that was described by Welch, et.al. in her paper Asbestos Exposure Causes Mesothelioma, but Not This Asbestos Exposure: An Amicus Brief to the Michigan Supreme Court, published in 2007 in the International Journal of Occupational and Environmental Health. In this paper, she identifies four questions that should be examined in the causation of disease in an individual:

1. Was the individual exposed to a toxic agent?
2. Does the agent cause the disease present in the individual?
3. Was the individual exposed to this substance at a level where the disease has occurred in other settings?
4. Have other competing explanations for the disease been excluded?

For question #2, there is ample literature that asbestos causes mesothelioma and no dispute in the medical literature. With respect to question #1, Mr. Rimondi had an exposure to asbestos from talcum powder for many years, fulfilling this criterion. Johnson's Baby powder has been shown to contain asbestos and Mr. Rimondi would have had asbestos exposure based on his descriptions from his deposition testimony and that of his family members. He has no other competing explanations (#4) for the development of her mesothelioma. The remaining criterion, #3 is whether there is an analogous exposure scenario in which others also developed mesothelioma. As described above, and recently

referenced by the Center for Disease Control, there are other individuals with exposure to contaminated talc products who then developed malignant mesothelioma.

Summary and Specific Causation in Mr. Rimondi's Case:

Based on the information that was provided to me and the diagnosis of mesothelioma as outlined by his treating physicians, and applying both my understanding of the medical literature and the facts of this case, it is my opinion to a reasonable degree of medical certainty that the exposures to the dust from asbestos-contaminated cosmetic talc products that Mr. Rimondi used for many decades, starting over 50 years ago, were above normal background levels. Both historic and recent analyses (published in the medical and scientific literature as well as industry, government and private laboratory testing) of the talc from the source mines used in Johnson's Baby Powder have shown significant amounts of chrysotile, anthophyllite, and tremolite asbestos. Similarly, studies of Johnson's Baby Powder talcum powders show asbestos contamination. Fiber release studies done recently by MVA Scientific Consultants and others from products using ore taken from the same source mines as those used in the manufacture of Johnson's Baby Powder showed significant amounts of chrysotile, anthophyllite, and tremolite asbestos. MSHA found anthophyllite in the mills that processed the Italian talc. Similarly, Dr. Compton found anthophyllite in 11 of the 13 samples of talc ore from the Italian mines, from which the talc originated that, was then used in consumer products. Studies to evaluate tremolite asbestos by Materials Analytical Services (MAS) showed that asbestos was detected in 17 of 30 samples of Johnson's Baby Powder evaluated. MAS performed an additional analysis to determine whether exposure of talcum powder below the waist led to respirable levels of asbestos (using Johnson and Johnson Baby Powder). Their report stated that there was a mean tremolite fiber exposure of 2.57 fibers/cc during this activity.

Mr. Rimondi's exposure to asbestos-contaminated talcum powders was the cause of his mesothelioma. If she had not used asbestos-containing talcum powder, Mr. Rimondi would not have developed malignant mesothelioma. Alternative powders not containing talc were available since the early 20th century.

The opinions related to Mr. Rimondi case are based on my review of the evidence of exposure in this case, the medical and scientific literature as described above regarding asbestos exposure and disease, available studies concerning fiber release, epidemiological studies of exposure to asbestos exposure and the development of disease, and my knowledge, skill, experience, and training as a physician specializing in occupational medicine with a clinical focus on evaluating individuals with asbestos exposure.

Mr. Rimondi has metastatic malignant mesothelioma of the pleura. His tumor was too far advanced when he presented to the doctor to be a surgical candidate. He has received different courses of chemotherapy, but has progressed. There is no cure for mesothelioma, and his prognosis is poor.

A list of references and reliance materials will be provided upon request.

Sincerely,

Jacqueline Moline, MD, MSc, FACP, FACOEM